U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 21 March 2007

IN THE MATTER OF:

W. B.,
Widow of J. B.,
Claimant,

v. Case No.: 2003-BLA-6262

CLINCHFIELD COAL CO.,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest.

DECISION AND ORDER ON REMAND – AWARD OF BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. §§ 901-945 and the regulations issued thereunder, which are found in Title 20 of the Code of Federal Regulations.

A Decision and Order Awarding Survivor's Benefits was issued by the undersigned Administrative Law Judge on October 13, 2004. The decision found that the weight of the evidence established that the miner's death was due to pneumoconiosis pursuant to 20 C.F.R. § 718.205(c). The decision was adjudicated under 20 C.F.R. Part 718 on the record pursuant to the parties agreement. The employer stipulated that the miner worked as a coal miner for eighteen years, that he had coal workers' pneumoconiosis and that the coal workers' pneumoconiosis arose from his coal mine employment.

The Benefits Review Board (BRB) in a Decision and Order dated November 3, 2005[1] vacated the decision and remanded the claim for a reevaluation of all relevant evidence because the decision provided no reason for according enhanced weight to the findings of the autopsy prosector, and because the decision did not explain how Dr. Joshua Perper's opinion concerning the miner's death was best supported by the record. The BRB also found that Dr. Perper's report contained no explanation or substantiation for a finding that clinical pneumoconiosis, standing alone, hastened death.

[1] The BRB did not return the record to the Office of Administrative Law Judges until March 9, 2006.

Claimant filed a November 30, 2005 report from Dr. Joshua Perper with Dr. Perper's curriculum vitae under cover of a letter dated March 1, 2006. The letter stated that Claimant was submitting the enclosures as evidence in this case. Respondent objected to the submission for reason that nothing in the BRB's decision directed that the record be reopened to allow for additional evidence and the Claimant did not file a motion or otherwise request that the record be reopened to received additional evidence. Respondent is correct. No order was issued allowing for additional evidence and no request to reopen the record to receive additional evidence was submitted. The Claimant's March 13, 2006 submittal is not considered.

Twenty C.F.R. § 718.205(c) provides that for the purpose of adjudicating claims after January 1, 1982, death will be considered to be due to pneumoconiosis where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by the complications of pneumoconiosis. Subsection (c)(5) of § 718.205(c) provides that pneumoconiosis is a substantially contributing cause of death if it hastens the miner's death.

The decision awarding benefits credited the report of Dr. Perper for the finding that the miner's death was substantially contributed to by coal workers' pneumoconiosis. It found that Dr. Perper's opinion on the cause of death was entitled to more credit than the opinions of Drs. Richard Naeye, P. Raphael Caffrey and James Castle because it is better supported by the record and is more in line with the findings of Dr. Larry Joyce, the autopsy prosector.

Dr. Perper wrote in the body of his report that "[c]oal workers' pneumoconiosis was a substantial contributory cause of Claimant's death both directly and indirectly through the associated centrilobular emphysema, that caused hypoxemia that either triggered or aggravated a fatal cardiac arrhythmia, and the complicating bronchopneumonia and pulmonary cancer." One of his report's three conclusions stated: "[c]oal workers' pneumoconiosis with associated centrilobular emphysema was a substantial contributory cause of [Claimant's] death and hastened his demise, both directly and through hypoxemia and complicating bronchopneumonia, and through the complicating carcinoma of the lung." Dr. Perper found the extent of the pneumoconiosis to be moderate to severe.

Dr. Perper's findings on causation are interpreted to mean that the Claimant's pneumoconiosis was severe enough to be a substantial contributory cause of death and hastened his demise through hypoxemia and complicating bronchopneumonia, as well as being the cause of his cancer. His findings are not interpreted to mean that pneumoconiosis can be considered a substantial contributor to the cause of death only if the pneumoconiosis can be considered the cause of the lung cancer.

Employer argues in its Brief on Remand and in its appeal to the BRB that Dr. Perper's opinion on the cause of the miner's death is speculative and unsupported by the record because the record contains no evidence of a fatal cardiac arrhythmia or hypoxemia caused by the Claimant's pneumoconiosis and/or emphysema. Employer is correct that the autopsy was performed only of the lungs, and thus Dr. Perper could not have examined the heart. However, a pathologist as well qualified as Dr. Perper is able to offer an opinion on how a disease process or

condition affects organs of the body without the opinion being considered to be speculative. Hypoxemia is deficient oxygenation in the blood. It is not unreasonable to appreciate that a severe pulmonary disease process would cause hypoxemia. The reports of the two pathologists and the pulmonologist offered by the Employer in response to the report by Dr. Perper do not dispute the finding of hypoxemia. Moreover, Dr. Perper also found that the pneumoconiosis hastened the death through complicating bronchopneumonia, and the existence of extensive acute bronchopneumonia is documented by the autopsy report as well as the report by Dr. Caffrey.

The difference in the opinions of the experts is not in the events of the pulmonary death, but in the etiology of the death, specifically whether the admittedly existing pneumoconiosis was severe enough to hasten the death. The root of Dr. Perper's finding that coal workers' pneumoconiosis had an affect on Claimant's death was that the pneumoconiosis was severe. It is also the source of the disagreement between him and Dr. Naeye on whether the pneumoconiosis hastened Claimant's death.

Severity of Pneumoconiosis

Dr. Naeye concluded that the pneumoconiosis was too mild to have hastened Claimant's death since it affected too little lung tissue to have any clinical significance. Yet, Dr. Naeye's reading of the microscopic tissue slides was the same as Dr. Perper in that Dr. Naeye identified the presence of moderately severe simple coal workers' pneumoconiosis. Their conclusions differed because Dr. Naeye expressed skepticism at the microscopic findings. He suggested that the tissue slides must not have represented the lungs as a whole. Dr. Naeye reported:

> The microscopic findings identify the presence of moderately severe simple coal workers' pneumoconiosis (CWP) in this man. These findings are not representative of his lungs as a whole. The cancer, its therapy and their resultant fibrosis and pneumonia caused such severe damage in his lungs that he would have died weeks earlier if the lung tissues available for my review were representative of his lungs as a whole. It is important to recognize that his CWP was not recognized on x-rays by multiple B-readers. [2]

Dr. Naeye seems to be saying that if the slides he observed were representative of the lungs as a whole, then the coal workers' pneumoconiosis would have substantially contributed to or hastened the death – "he would have died weeks earlier" - the conclusion reached by Dr. Perper.

If Dr. Perper's conclusion is credited, the pneumoconiosis must be considered to have been severe and thus to have substantially contributed to or hastened the death. It follows that if Dr. Naeye's conclusion is credited, the pneumoconiosis will be considered to have affected too little lung tissue to have any clinical significance. The determination depends on whether the microscopic tissue slides read by the pathologists were representative of the lungs as a whole. The Decision and Order Awarding Survivor's Benefits determined that the report of Dr. Perper

[2] May 15, 2002 report of Dr. Richard Naeye, p. 2.

should be credited because it is better supported by the record as it is more in line with the findings of Dr. Joyce, the autopsy prosector, as Dr. Joyce observed severe and diffuse pneumoconiosis with coal macules in all lobes. The BRB's Order of Remand disagreed with this analysis, stating that the Decision and Order Awarding Survivor's Benefits "provided no reason for according enhanced weight to the findings of the autopsy prosector; moreover, Dr. Joyce did not address the cause of the miner's death, but merely supported Dr. Perper's opinion regarding the degree of severity of the miner's pneumoconiosis."

The reasoning behind giving enhanced weight to Dr. Joyce's observation of severe and diffuse pneumoconiosis with coal macules in all lobes, an important corroboration of the findings of Dr. Perper, is because, as the autopsy prosector and the pathologist responsible for preparation of the tissue slides, he was be in the best position to know whether the slide tissues were representative. Dr. Joyce did not address the cause of death, but his corroboration of the severity of the pneumoconiosis addresses the basis of Dr. Perper's finding on the cause of death, and it addresses the reason Dr. Naeye gave for finding that the pneumoconiosis could have had no affect on the death.

Dr. Naeye's reasoning for dismissing the tissue slides he observed as not representative of the lungs as a whole included his finding that "his CWP was not recognized on x-rays by multiple B-readers." But those x-rays were taken from 1880 through November of 1991, the most recent being taken about nine years before the Claimant's death. As pneumoconiosis is a progressive disease, the difference between the x-ray readings and the autopsy results is indicative of the progress of the disease. It is undisputed that the disease progressed to at least the presence of pneumoconiosis. A problem with Dr. Naeye's analysis here is that he does not believe that pneumoconiosis is progressive. His May 15, 2002 report reads at p. 2: "…there is strong evidence that CWP does not progress after a miner leaves exposure to coal mine dust."

Dr. Naeye also reports that he considered the interpretations of fifteen lung x-rays taken in the year 2000 and none mention the presence of lesions that suggest pneumoconiosis. However, those x-rays would have been taken during Claimant's treatment for terminal lung cancer. It is not surprising that they would not have been read for lesions indicating pneumoconiosis.

Dr. Raphael Caffrey read the tissue slides as showing less extensive pneumoconiosis than either Dr. Perper or Dr. Naeye, as he reported finding mild to moderate pneumoconiosis with focal micronodules. He expressed disagreement with Dr. Perper's position, which he interpreted as a finding that Claimant's pneumoconiosis caused the centrilobular emphysema, hypoxemia, fatal cardiac arrhythmia and pulmonary cancer. Other than citing studies discussing the possibility of coal dust causing cancer, Dr. Caffrey did not explain his finding that the Claimant's pneumoconiosis did not contribute to death. Dr. Caffrey merely offered the opinion that the conditions resulting in Claimant's death were the result of years of cigarette smoking.

Employer also offered the report of Dr. James Castle, a pulmonary specialist. Dr. Castle reported that Claimant had pathologic evidence of pneumoconiosis, but that it had no affect on his death. Dr. Castle's report concentrated on the cause of Claimant's lung cancer. He reasoned that the miner's carcinoma of the lungs could not have been caused by coal dust as there was no

finding of silicosis, and, in his opinion, there is no medical support for the proposition that coal dust exposure can cause lung cancer. Dr. Castle also commented on the severity of the Claimant's pneumoconiosis, as he contended that it was not serious enough to cause radiographic abnormalities. His opinion was based on his review of the record showing chest x-rays that do not reveal opacities. Again, the chest x-rays referenced by Dr. Castle were taken between 1980 and 1991, with the most recent being taken some eight or nine years before the Claimant's death. Dr. Castle's opinion of the severity of the Claimant's pneumoconiosis based on his reference to these x-ray readings is clearly not as creditable as the findings by autopsy.

The BRB's Order also requires that conflicting evidence be weighed and findings of fact be rendered as to the etiology of the miner's emphysema and lung cancer. As previously stated, Dr. Perper opined that the pneumoconiosis with associated emphysema was a substantial contributory cause of death. He also found that the lung cancer was caused by exposure to coal dust and smoking.

Centrilobular Emphysema

Dr. Perper reported finding moderate to severe centrilobular emphysema that was caused by exposure to coal mine dust and cigarette smoking. In support of his finding of coal dust exposure being a causative factor, he referenced scientific literature as stating that coal dust exposure has a significant role in causing centrilobular emphysema beyond any effect that may be attributed to smoking. He offered that no logical reason exists here to exclude coal dust exposure as a cause of the centrilobular emphysema. In fact, Dr. Perper refers to Claimant's coal dust related pulmonary condition as "coal workers' pneumoconiosis and associated centrilobular emphysema."

Dr. Naeye's findings on the presence and affect of centrilobular emphysema here are internally inconsistent. In his May 15, 2002 report he interpreted the tissue slides as showing moderately severe centrilobular emphysema and noted that "[i]n the few lung areas where cancer and its consequences were mild, centrilobular emphysema and chronic bronchitis were the only disorders that could have measurably affected lung function." However, in a follow up report dated December 26, 2002, Dr. Naeye states that "there is no clinical evidence that this man had clinically significant centrilobular emphysema.

As to the etiology of the emphysema, Dr. Naeye states that coal dust rarely has a significant role in the genesis of centrilobular emphysema. He cites two studies for the proposition that coal mine dust has no effect on life expectancy when cigarette smoking is taken into consideration. He extrapolates from those studies the conclusion that such life expectancy surely would be shortened if coal dust exposure had a significant role in the genesis of centrilobular emphysema.

The regulations implementing the Black Lung Benefits Act, were amended, effective January, 2001. The amendments included a revision to the definition of pneumoconiosis at 20 C.F.R. § 718.201 by inserting the terms "legal" and "clinical" pneumoconiosis. The stated reason for the change was to clarify that both restrictive and obstructive lung disease may fall within the definition of pneumoconiosis, and to recognize the latent and progressive nature of the

disease. The preamble to the regulations explained the medical reasons for revision. The explanation included studies such as *Post-mortem study of emphysema in coal workers and non-coal workers* which showed that "centrilobular emphysema (the predominant type observed) was significantly more common among the coal workers. The severity of the emphysema was related to the amount of dust in the lungs. These findings held even after controlling for age and smoking habits." It also quotes a study by Leigh J, Outhred KG, McKenzie HI, Glick M, Wiles AN, *Qualified pathology of emphysema, pneumoconiosis and chronic bronchitis in coal miners* BR J Indus Med 40:258-263 (1983) "The authors concluded that 'these results provide strong evidence that emphysema in coal workers is causally related to lung coal content.'" Fed. Reg. Vol. 65, No. 245, Dec. 20, 2000, p. 79941.

The aforesaid studies reviewed and cited with approval by the Department of Labor are contrary to Dr. Naeye's contention that centrilobular emphysema can not be caused by coal dust exposure. Thus Dr. Naeye's opinion on the etiology of the Claimant's centrilobular emphysema is not credited.

Dr. Caffrey diagnoses a moderate degree of centrilobular emphysema. His diagnosis is consistent with the diagnosis of Dr. Perper of moderate to severe centrilobular emphysema. He disagrees with Dr. Perper's opinion that the centrilobular emphysema was caused by coal dust exposure, but he provides no reason for his disagreement other than to observe that smoking is the number one cause of centrilobular emphysema. There is no dispute that Claimant had a significant smoking history and that his cigarette smoking was likely a cause of his centrilobular emphysema. However, cigarette smoking and coal dust exposure are not mutually exclusive causative factors. At issue is whether his coal dust exposure was also a causative factor, and Dr. Caffrey's report does not speak to that question.

Dr. Castle reported that his review of the record reveals evidence of pulmonary emphysema, and that Claimant had a sufficient smoking history to cause "chronic bronchitis/emphysema." He found that Claimant had a sufficient exposure to coal dust for him to develop coal workers' pneumoconiosis; however, he provides no opinion on whether the coal dust exposure could have caused the pulmonary emphysema.

Accordingly, Dr. Perper's conclusion that coal workers' pneumoconiosis with associated centrilobular emphysema was a substantial contributory cause of Claimant's death and hastened his demise is accepted.

Lung Cancer

Dr. Perper referred to what he called a growing body of scientific medical literature substantiating a causal connection between exposure to coal mine dust containing silica and the development of lung cancer. He explained that the International Association in Cancer Research and OSHA have recognized in recent years that silica is carcinogenic in humans and workers' exposed to silica can contract cancer of the lungs and that coal workers are definitely exposed to coal dust containing silica.

Drs. Naeye and Caffrey disagree with Dr. Perper on the etiology of the lung cancer. They argue that there is no evidence that the squamous cell carcinoma could have been caused by coal dust exposure. They attributed its cause to cigarette smoking.

Dr. Perper explained that cancer has been related in recent years to occupational exposure to silica, that silica has been recognized in recent years to be carcinogenic in humans and that numerous collections of silica crystals were seen in Clamant's lung sections. Dr. Naeye answers that Dr. Perper does not differentiate between non-toxic silicates which are plentiful and easily recognizable in the lungs of Claimant and toxic free silica crystals which are relatively few in numbers. Dr. Caffrey also reported that he saw very few crystals which were consistent with silica. Also, Dr. Castle pointed out that none of the pathologists diagnosed silicosis and he emphasized Dr. Naeye's and Dr. Caffrey's findings that the vast majority of bifringement crystals were due to silicates which are non-toxic.

Drs. Naeye and Caffrey argue persuasively that the studies on mortality of coal workers from lung cancer show that coal miners do not suffer an increase in lung cancer when cigarette smoking is taken into consideration, and there would be an increase of carcinoma in bituminous coal workers' lungs if the silica observed by Dr. Perper is carcinogenic. Dr. Naeye references testimony by the Assistant Attorney General of the United States and reports by the World Health Organization concluding that exposure to coal dust does not increase the frequency of lung cancer.

Accordingly, the record does not support a finding that the squamous cell carcinoma was caused by coal dust exposure. As stated by Dr. Caffrey, Dr. Perper does not take into account the aforesaid studies on whether coal dust is carcinogenic.

However, a weighing of the evidence does support Dr. Perper's finding that Claimant's moderate to severe coal workers' pneumoconiosis was caused by coal dust exposure and his moderate to severe centrilobular emphysema was caused by coal dust exposure. Thus, Dr. Perper's finding that the "coal workers' pneumoconiosis with associated centrilobular emphysema was a substantial contributory cause of death and hastened death through hypoxemia and bronchopneumonia" is credited. The contrary opinions do not take into consideration that the associated centrilobular emphysema was caused by exposure to coal dust. None provide an opinion inconsistent with Dr. Perper's finding that the centrilobular emphysema was a substantial contributor to death.

Accordingly,

ORDER

IT IS ORDERED that the survivor's claim for benefits filed by WB is hereby granted.

IT IS FURTHER ORDERED that, within 30 days of the date of issuance of this *Decision,* Claimant's counsel shall file, with this Office and with opposing counsel, a petition for

a representative's fees and costs in accordance with the regulatory requirements set forth at 20 C.F.R. § 725.366 (2005). Counsel for Employer shall file any objections with this Office and with Claimant's counsel within 20 days of receipt of the petition for fees and costs. It is requested that the petition for services and costs clearly provide (1) counsel's hourly rate with supporting argument or documentation, (2) a clear itemization of the complexity and type of services rendered, and (3) that the petition contains a request for payment for services rendered and costs incurred before this Office only as the undersigned does not have authority to adjudicate fee petitions for work performed before the district director or appellate tribunals. *Ilkewicz v. Director, OWCP*, 4 B.L.R. 1-400 (1982).

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Thomas M. Burke
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is:

Benefits Review Board
U.S. Department of Labor
P.O. Box 37601
Washington, DC 20013-7601

Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481. If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).